

Mineral trioxide aggregate (MTA) and calcium hydroxide as pulp-capping agents in human teeth: a preliminary report

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Abstract

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Aim To compare mineral trioxide aggregate (MTA) with calcium hydroxide when used as pulp-capping materials in human teeth.

Methodology Eleven pairs of maxillary third molars in subjects between 20 and 25 years of age were subjected to mechanical pulp exposure. The exposed pulps were capped with MTA or calcium hydroxide, covered with ZOE and restored with amalgam. A total of 14

teeth were extracted after periods of 1 week (two molars), 2 months (three molars), 3 months (five molars), 4 months (two molars) and 6 months (two molars).

Results Histological evaluation demonstrated less inflammation, hyperaemia and necrosis plus thicker dentinal bridge and more frequent odontoblastic layer formation with MTA than calcium hydroxide.

Conclusions Although the results favour the use of MTA, more studies with larger samples and a longer follow up are suggested.

Keywords: calcium hydroxide, mineral trioxide aggregate (MTA), pulp cap.

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Introduction

The procedure of pulp capping relies primarily on the ability of pulpal tissue to heal. Various factors affect this process including age, periodontal condition and stage of root formation (Camp *et al.* 2002). Procedural factors such as the size of exposure, its nature (traumatic, mechanical or carious) and microbial contamination of the site have also been described as determinants of the success of pulp capping (Camp *et al.* 2002). However, the importance of these factors has been challenged (Stanley 1989).

A wide array of materials have been used for pulp capping, but calcium hydroxide remains the standard

(Camp *et al.* 2002). Subsequent to pulp capping with this conventional alkaline agent, the adjacent pulp tissue is usually completely deranged and distorted, forming a *zone of obliteration*. A weaker chemical effect on the sub-jacent, more apical tissue results in a *zone of coagulation necrosis*. This layer causes sufficient stimulation to the vital pulp tissue to respond (Stanley 1989). Some question the superiority of calcium hydroxide, because of its degradation over time, tunnel defects through dentinal bridges under it and poor sealing properties (Schuurs *et al.* 2000).

Mineral trioxide aggregate (MTA) has been used in pulp-cap procedures in animals, demonstrating remarkable success compared with calcium hydroxide (Abedi *et al.* 1996, Pitt Ford *et al.* 1996, Junn *et al.* 1998, Faraco & Holland 2001). However, no studies have so far evaluated MTA as a pulp-capping agent in humans. The purpose of this study was to compare the properties of MTA with calcium hydroxide in human tooth pulp-capping treatment.

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Table 1 Categorization of inflammation and hyperaemia

Absent (0)	Mild (1)	Moderate (2)	Severe (3)	
Inflammation	No inflammatory cells	<30 inflammatory cells	≤30 inflammatory cells <60 inflammatory cells	≤60 inflammatory cells
Hyperaemia	1 vessel <3 vessels	≤3 vessels <5 vessels	≤5 vessels <8 vessels	≤8 vessels

Materials and methods

Twenty-two intact maxillary third molars free of restorations, from subjects between 20 and 25 years of age which required extraction were selected. Percussion and pulp-sensitivity tests were performed and radiographs were examined to assess pulpal health. Only those teeth that could be extracted without surgery and with minor trauma were included. All subjects were informed of the possible complications of the procedure. Ethical approval was sought and granted. Informed consent was obtained from all subjects.

Lidocaine was used to anaesthetize the teeth. They were subsequently isolated with rubber dam and a conventional Class I cavity of approximately 1 mm width was prepared on the occlusal surface. A standard exposure of 0.5 mm diameter was created in the pulp with a high-speed handpiece and 005 round bur by an operator who did not have prior knowledge of the pulp-cap agent to be used. No salivary contamination was allowed. Homeostasis was gained by irrigating the cavity with sterile saline and application of small pieces of cotton before capping with calcium hydroxide (Dycal[®], L.D. Caulk, Milford, DE, USA) or MTA (ProRoot[®], Dentsply Tulsa, Tulsa, OK, USA) on the contralateral third molars of the same subject.

Using a stiff metal spatula, MTA powder was mixed with saline in a 3 : 1 ratio and then placed over the exposure site with a plastic instrument. Calcium hydroxide paste was mixed and applied to the exposure site with a plastic instrument. Zinc oxide–eugenol (ZOE) cement at a thickness of 2 mm was used over both materials. Amalgam served as the filling material.

A total of 14 teeth were extracted after periods of 1 week (two molars), 2 months (three molars), 3 months (five molars), 4 months (two molars) and 6 months (two molars). The apex of each tooth was immediately sectioned to allow penetration of 10% formalin for tissue fixation purposes. The teeth were kept in formalin for 1 week and subsequently decalcified in 10% formic acid for 2–3 weeks. Samples were exposed to ascending concentrations of alcohol (70, 90 and 100%), cleared in methyl salicylate and submerged in paraffin for 12 h.

Six-micrometre sections were cut in a buccolingual direction every 100 µm. Haematoxylin and Eosin (H&E) staining was used.

An oral and maxillofacial pathologist studied the images taken from slides under a microscope without knowledge of the source of specimens. Hyperaemia and inflammation were recorded under a magnification of ×312.5 and categorized as shown in Table 1.

Dentine bridge thickness was measured to 10 µm accuracy using computer software (Adobe Photoshop 6). The true thickness of the dentine bridge formed was registered in millimetres. Every sample was also evaluated for severity, type and site of inflammation, presence of necrosis, hyperaemia, calcification other than in the area of the bridge and odontoblastic layer.

Results

Tables 2 and 3 outline the length of the specific time intervals. Following the initial procedure, two subjects did not return for extraction and two other subjects agreed to the extraction of only one tooth; two teeth were lost in the process of histological preparation. The number of samples evaluated was 14.

Samples capped with mineral trioxide aggregate

One-week sample: No bridge was formed and mild chronic inflammation at the exposure site plus slight hyperaemia and necrosis were observed. Lymphocytes and few polymorphonuclear leucocytes (PMNLs) were seen.

Two-month samples: Dentine bridges of maximum 0.28 mm thickness with underlying mild chronic inflammation were found (Fig. 1). Lymphocytes dominated the inflammatory zone. Mild hyperaemia and necrotic areas could be seen in only one sample. The other sample had calcifications and a few odontoblasts.

Three-month samples: Dentine bridges of maximum thickness of 0.25 mm and odontoblasts were registered for all samples (Fig. 2). Mild chronic inflammation, mild hyperaemia and necrosis were observed underneath the bridge of one sample. Calcifications and dominating lymphocytes also could be seen.

Table 2 Samples capped with MTA

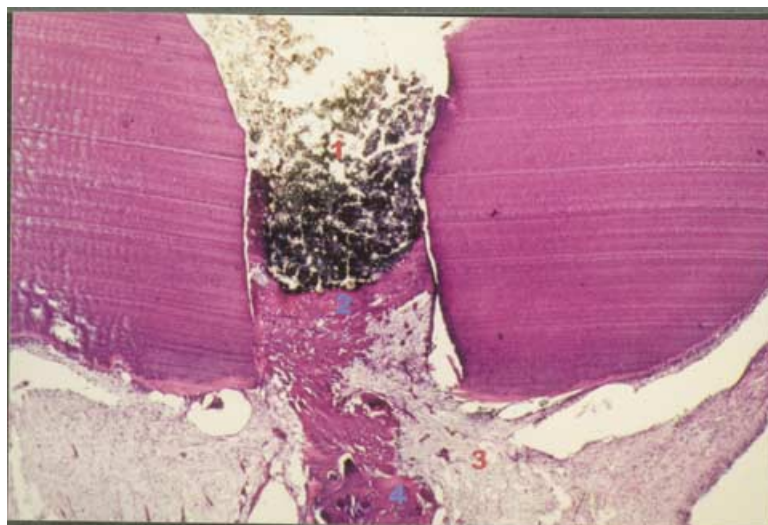
Time interval	Odontoblastic layer	Calcification*	Hyperaemia	Necrosis	Inflammation			Thickness (mm)	Dentinal bridge	Sample number
					Site	Type	Degree			
One week	–	–	1	+	Under exposure area	Chronic	1	–	–	1
Two months	–	–	1	+	Under bridge	Chronic	1	0.12	+	2
Two months	+	+	0	–	Under bridge	Chronic	1	0.28	+	3
Three months	+	+	1	+	Under bridge	Chronic	1	0.25	+	4
Three months	+	–	0	–	–	–	0	0.19	+	5
Three months	+	–	0	–	–	–	0	0.25	+	6
Four months	–	–	0	–	–	–	0	0.37	+	7
Six months	+	–	0	–	–	–	0	0.43	+	8

*Other than in the area of the dentinal bridge.

Table 3 Samples capped with calcium hydroxide

Time interval	Odontoblastic layer	Calcification*	Hyperaemia	Necrosis	Inflammation			Thickness (mm)	Dentinal bridge	Sample number
					Site	Type	Degree			
One week	–	–	1	+	Under exposure area	Acute-Chronic	2	–	–	9
Two months	–	+	2	+	Coronal pulp	Acute-Chronic	3	–	–	10
Three months	–	–	1	+	Under bridge	Chronic	2	0.02	+	11
Three months	–	–	1	+	Under bridge	Chronic	2	0.02	+	12
Four months	–	–	1	–	Under bridge	Chronic	1	0.04	+	13
Six months	–	+	1	+	Under bridge	Chronic	1	0.15	+	14

*Other than in the area of the dentinal bridge.

**Figure 1** A view of the pulp cap area of a 2-month sample capped with MTA, H&E stained, $\times 31.25$. (1) MTA; (2) dentinal bridge; (3) pulp; (4) calcification.

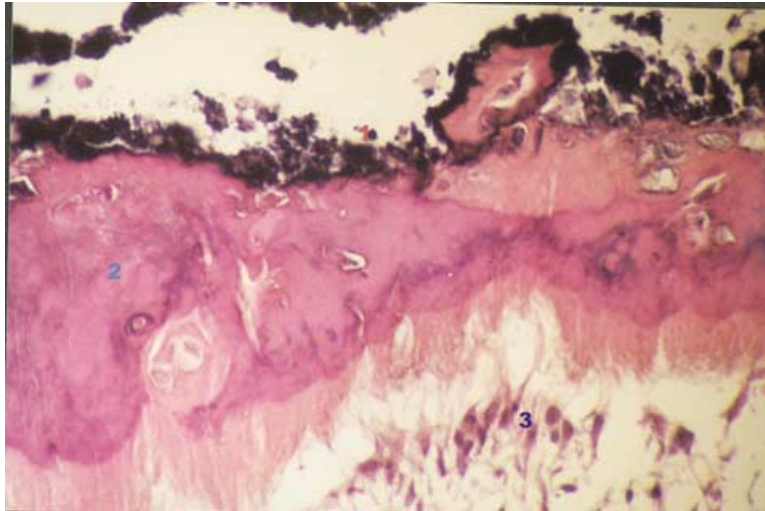


Figure 2 A view of the pulp cap area of a 3-month sample capped with MTA, H&E stained, $\times 312.5$. (1) MTA; (2) dentinal bridge; (3) odontoblasts.

Four-month sample: Dentinal bridges of 0.37 mm thickness were seen. Inflammation, hyperaemia, necrosis, calcification or odontoblastic layer were not observed.

Six-month sample: A 0.43 mm-thick dentine bridge and a nearly regular odontoblastic layer were noted. No inflammation, necrosis or calcifications were registered.

Samples capped with calcium hydroxide

One-week sample: No dentinal bridges were present. Chronic and acute inflammation at the exposure site and hyperaemia were noted. PMNLs and to a lesser extent lymphocytes were the dominant inflammatory cells. There were no calcifications.

Two-month sample: No dentine bridges had formed. Acute and chronic inflammation, hyperaemia and calcifications were prominent with dominating PMNLs and lymphocytes.

Three-month samples: Both samples showed moderate chronic inflammation, mild hyperaemia and necrosis. Lymphocytes were the dominant inflammatory cells (Figs 3 and 4). An irregular bridge of maximum 0.02 mm thickness was reported. No calcifications or odontoblastic layers could be seen.

Four-month sample: A bridge of maximum 0.04 mm thickness had formed with mild chronic lymphocyte-dominated inflammation and mild hyperaemia underneath. No calcifications or odontoblastic layer were present.

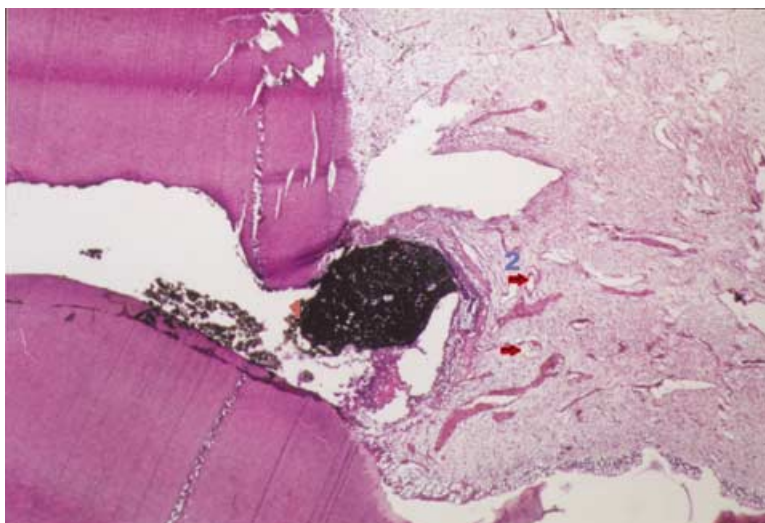
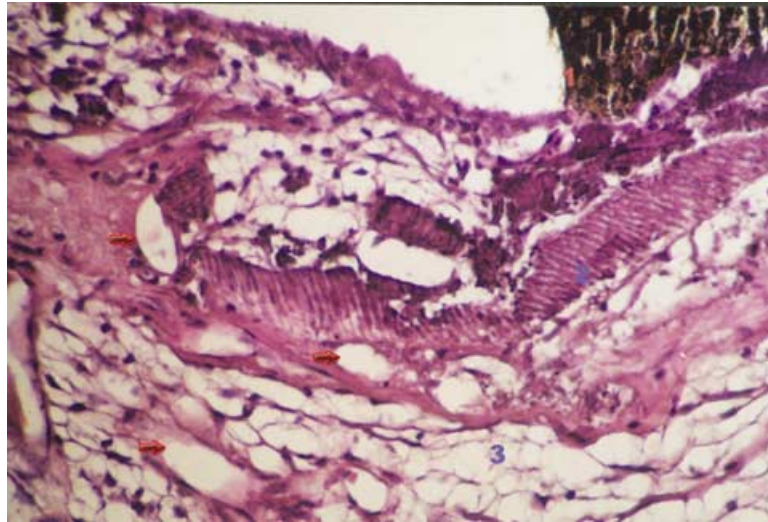


Figure 3 A view of the pulp cap area of a 3-month sample capped with calcium hydroxide, H&E stained, $\times 31.25$. (1) Calcium hydroxide; (2) pulp. A few vessels can be seen (arrows) indicating mild hyperaemia.

Figure 4 A view of the pulp-cap area of a 3-month sample capped with calcium hydroxide, H&E stained, $\times 312.5$. (1) calcium hydroxide; (2) dentine-like structures; (3) pulp. Polymorphonuclear leucocytes dominate the superficial layers (top), whereas lymphocytes are more frequently dispersed in the deeper parts (bottom). Mild hyperaemia is also present (arrows).



Six-month sample: Calcification and necrosis were seen underneath a bridge of maximum 0.15 mm thickness. No odontoblastic layer was showed. Lymphocytes dominated the mild chronic inflammation and there was mild hyperaemia.

Discussion

A dentinal bridge may be a sign of healing or irritation (Schuurs *et al.* 2000) and it is known that the presence of bacteria is a significant factor in inhibiting healing of pulp exposures (Watts 1979, Browne *et al.* 1983, Cox 1987). Unfortunately, calcium hydroxide does not adhere to dentine and lacks the ability to seal. Tunnel defects in dentine bridges under calcium hydroxide dressings can act as pathways for microleakage (Cox *et al.* 1985). This material also has a tendency to dissolve over time (Schuurs *et al.* 2000).

Although a statistical analysis was not performed in this study, due to the small number of samples, the results show that inflammation was seen more frequently and with a greater severity in samples capped with calcium hydroxide. With MTA, thicker bridges formed and the presence of an odontoblastic layer was a frequent finding. In addition, few samples capped with MTA had hyperaemia, whereas hyperaemia was seen in every sample capped with calcium hydroxide, and virtually no odontoblastic layer was formed. Necrosis was also a more frequent finding with calcium hydroxide. It should be emphasized, however, that these results are relevant for only small exposures created mechanically and without caries.

Cox *et al.* (1985) and Cox (1987) used ZOE to prevent bacterial leakage as in the present study, so that inferior results with calcium hydroxide compared to MTA cannot be attributed to leakage. Staining bacteria in histological sections is difficult and may not show bacteria despite their presence in the samples (Mjör 1977). In this study, no sections were stained for bacterial invasion of the site.

Abedi *et al.* (1996) found a significantly higher frequency of calcific bridge formation and less inflammation with MTA compared with calcium hydroxide in an animal study. Pitt Ford *et al.* (1996) also showed dentine bridge formation in all pulps capped with MTA and no inflammation except in one sample. In contrast, all samples capped with calcium hydroxide preparation showed pulpal inflammation, and bridge formation occurred in only two samples. In accordance with these and the present study, Junn *et al.* (1998) reported significant differences between the amount of inflammation and the degree of dentinal bridge formation in the MTA and calcium hydroxide groups of 63 teeth from four Beagle dogs.

The ability of MTA to induce the formation of a dentine bridge may be due to its excellent sealing ability (Torabinejad *et al.* 1993, 1994, Bates *et al.* 1996, Fischer *et al.* 1998, Wu *et al.* 1998) or biocompatibility (Kettering & Torabinejad 1995, Torabinejad *et al.* 1997, 1998, Holland *et al.* 1999, Mitchell *et al.* 1999, Keiser *et al.* 2000). MTA can induce cytokine release from bone cells (Koh *et al.* 1995, 1997, 1998) and can allow the attachment of osteoblasts in the form of a monolayer (Zhu *et al.* 2000); however, some studies suggest that it is only osteoconductive and not osteoinductive (Moretton *et al.* 2000). It has suf-

ficient compressive strength to allow condensing of amalgam and a negligible solubility (Torabinejad *et al.* 1995).

However, Myers *et al.* (1996) found no significant differences in pulpal status or bridging between MTA and calcium hydroxide groups of their study on dogs. They concluded both MTA and calcium hydroxide performed equally well as pulp-capping agents. It has not been possible to identify whether the inductive effects of calcium hydroxide are due to release of calcium or hydroxyl ions (Schubich *et al.* 1978) and early work has suggested calcium ions are not necessary for the repair process (Glass & Zander 1949, Seltzer & Bender 1958). Despite many studies, the mechanism of action of this material remains unknown (Pashley 1996).

Faraco & Holland (2001) emphasized the advantages of MTA over calcium hydroxide for pulp capping. Thirty teeth of three dogs were capped with either calcium hydroxide or MTA. More inflammation and less frequent dentinal bridging were observed in the calcium hydroxide group in addition to material resorption and microleakage of microorganisms. However, a subjective method of evaluating inflammation had been employed.

Conclusion

In light of the results of the present and other relevant studies, MTA is superior to calcium hydroxide for pulp capping of mechanically exposed human teeth. However, further research with larger samples and a longer follow up is warranted.

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